University of Toronto
Library
Serials Dept.
Toronto, Ont. M5S 1A5

MEDICAL JOURNAL

University of Toronto

medical journal
university of toronto

Established December 1923
An Undergraduate Publication

Volume LV
Number 5

STAFF

EDITOR-IN-CHIEF: Susan Ross, 7T9

ASSOCIATE EDITORS: Molly Wills, 7T9
Felix Tyndel, 7T9
David Arnold, 7T9
Tina Feirabend, 8T1
Peter Hayashida, 8T1
Lin Leung, 8T1
Lin Tsai, 8T1
Les Urbankiewicz, 8T1
Ken Zatzman, 8T1

ART, GRAPHICS, & COVER: Arts as Applied to Medicine,
Jackie Heda, liason

CONTENTS

Common Low Back Pain	**91**
Medline	**97**
Partial Meniscectomy	**100**
Thin Air	**106**
Social Policy for Mental Health in Ontario	**110**

PATRONS

Each year a number of our supporters donate an amount well above and beyond the mere subscription rate. For their generous financial support [with its consequent psychological boost] we would like to extend our sincere thanks to the following individuals:

Dr. E.C. Allen, Toronto
Dr. Ulo Ambus, Toronto
Dr. Crawford S. Anglin, Toronto
Dr. P.W. Alberti, Toronto
Dr. R.J. Angus, Toronto
Dr. D.C. Arnot, Don Mills
Dr. Michael A. Baker, Toronto
Dr. B. Lynn Beattie, Toronto
Dr. W. Douglas Bennett, Toronto
Dr. John A. Beswick, West Hill
Dept. of Nutrition & Food Science
Dept. of Surgery, U. of T.
Dr. Robert E. Ecclestone, Thornhill
Dr. Harold E. Edwards, Toronto
Eli Lilly & Co. (Canada) Ltd.
Dr. Emmanuel Farber, Toronto
Dr. W.H. Francombe, Toronto
Dr. Frieda Fraser, Burlington
Dr. John Fredrickson, Toronto
Dr. Armis Freiberg, Toronto
Dr. A. Geisler, Mississauga
Dr. Melvyn Goldberg, Toronto
Dr. Irvin Gollish, Toronto
Dr. D.A. Gordon, Toronto
Dr. Milton M. Green, Michigan
Dr. L.F.L. Grover, Islington
Dr. Abraham L. Halpern, N.Y.
Dr. Robert Hicks, Toronto
Dr. F. Marguerite Hill, Toronto
Dr. Irwin M. Hillard, West Hill
Dr. R.B. Holmes, Toronto
Dr. W.J. Horsey, Toronto
Dr. Mary Jackson, Toronto
Dr. F.L. Johnson, Hamilton
Dr. H.B. Kedward, Toronto
Dr. James H. Kerr, Don Mills
Dr. H.F.King, Midland
Dr. R.G. Kunkel, Noranda, Quebec
Dr. Bernard Langer, Toronto
Dr. Jack R. Leitch, Toronto
Dr. William J. Lindsay, Toronto
Dr. Kenneth Livingston, Toronto
Dr. Lynn F.W. Loach, Toronto
Dr. Alan Lyall, Toronto
Dr. Robert Mackenzie, Toronto
Dr. C. Ross MacKay, Toronto
Dr. L.W. MacPherson, Toronto
Dr. Keith L. Moore, Toronto
Dr. Bruce Marshall, St. Marys
Medical Alumni Association
Dr. James F. Murray, Toronto
Dr. Dimitrios Oreopoulos, Toronto
Dr. R.H. Penney, Toronto
Dr. R.L. Perkin, Mississauga
Dr. Mel E. Petersiel, Scarborough
Dr. C.P. Rance, Toronto
Dr. W.J. Reynolds, Toronto
Dr. J.H. Rickard, Toronto
Dr. R.K. Schacter, Toronto
Dr. D.A. Stewart, Toronto
Dr. Ron Taylor, Toronto
Dr. D. Vaughan, San Jose, Calif.
Dr. Robert Volpe, Toronto
Dr. Paul G. Walfish, Toronto
Dr. J.E. Weir, Halifax
Dr. George E. Wodehouse, Toronto
Dr. M.S. Wolochow, Vancouver
Dr. C.R. Woolf, Toronto

GUIDELINES FOR PUBLICATION

1) All articles intended for publication should be submitted to the editors before the deadline which is usually *six* weeks before the release of the Journal.
2) Submitted articles should be typed (or legibly printed) double-spaced; single-sided on paper 8½" x 11" (letter size) and they should not be more than 15 to 20 pages (3000 to 4000 words).
3) Illustrations, graphs, diagrams and figures should be put on a separate sheet and identified by Arabic numerals. A list of explanatory legends for all the figures and diagrams should be put on another sheet. Do not write on the back of photographs.
4) References should be checked with the original sources, designated by Arabic numerals in the text, numbered in order of appearance and put on a separate sheet. Each periodical reference should include in order: the author's name and initials, title of the article, Journal name, volume number, page number and year. (eg., 1. Rosen HJ: Lumbar invertebral disc surgery: review of 300 cases. *Can. Med Assoc J., 101: 317, 1969.*)
5) The editors reserve the right to make such editorial changes as are deemed necessary to ensure correctness of grammar and spelling, clarification of statements and conformity to Journal style.
6) Acceptance for publication is subject to the understanding that such material is submitted solely to the Journal and will not be reprinted without the consent of both the editors and author.

Common Low Back Pain

K.H. Chan

Lover's Parting Such Public Sorrow

LONDON (Reuter) - Wedged into a two-seater sports car, a near-naked man was suddenly immobilized by a slipped disc, trapping his woman companion beneath him, according to a doctor writing in a medical journal here.

The desperate woman tried to summon help by honking the horn with her foot. A doctor-ambulanceman, fireman, and a group of interested passers-by quickly surrounded the couple's car in Regents Park.

"The lady found herself trapped beneath 200 pounds of pain-racked, immobile man", said Dr. Brian Richards of Kent.

"To free the couple, firemen had to cut away the car frame", he added.

The distraught woman, helped out of the car and into a coat, sobbed, "How am I going to explain to my husband what has happened to his car?"

October 1977

Low back pain is a common condition seen by the family physician. It is estimated that 65% of the population will be afflicted with significant low back pain at one time or another during their life time. Because of its frequency of recurrence and its disabling effects, correct diagnosis and appropriate treatment based on an understanding of the underlying condition is of utmost importance.

Too often the uncommon and potentially serious causes of low back pain are emphasized in medical school to the neglect of the overwhelmingly common causes of low back pain seen in general practice. To put things in the proper perspective the great majority of low back pains are either discogenic pain or facet joint pain. Only a small proportion of the discogenic pain, perhaps less than 10%, has disc herniation with nerve root compression.

For the sake of completeness and in case one should fall into a diagnostic trap as a result of complacency, it is helpful to use the following classification[1] to arrive at the appropriate differential diagnoses:

Classification	Examples of Lesions
1) Viscerogenic -	genito-urinary tract and pelvic organs.
2) Vasculogenic -	aortic aneurysm.
3) Neurogenic -	infections and neoplasms of spinal cord or cauda equina.
4) Spondylogenic -	a) bony lesions b) soft tissue lesions - disc lesions - facet joint lesions.
5) Psychogenic -	probably **no** back pain is purely psychogenic; an element of organic pain is present in every functional back pain.

Pathogenesis

Discs

Disc degeneration is an age related process whereby there is a gradual loss of the mucopolysaccharides with a decrease in the fluid content of the nucleus pulposus. This results in loss of turgor, elasticity and thickness of the discs. The annulus fibrosus also loses some of its elasticity with age. The fibres become more easily separated and torn permitting herniation especially at its posterior aspects.

The exact etiology of the pain producing mechanism has not been definitively elucidated. The following are the possible pain-provoking mechanisms:

1) Chemicals derived from the breakdown of the mucopolysaccharides seep through the cracks in the annulus to provoke pain.

2) Inflammatory response to herniated disc material. The inflammatory response may be autoimmune in nature.

3) Inflammatory response to mechanical injury of the disc(such as tears in the annulus).

4) Bulges in annulus due to weakening, stretch the nerve endings over the surface.

5) Herniations of disc material likewise distort the nerve endings.

However, it must be noted that disc degeneration may occur and may remain asymptomatic.

Facet Joints

The facet joints are about the size of inter-phalangeal joints. They are diarthrodial joints with cartilagenous articular surfaces, synovium and capsular ligaments. These joints were thought to be non-weight bearing whose sole function was to guide, steady and limit the movements of the vertebral bodies. At the levels of the secondary spinal curves in the cervical and lumbar regions, the weight-bearing function of the facet joints is revealed by the presence of meniscoids along the dorsal and ventral surfaces of the joints. Especially in the lumbar region, the facet joints are more prone to osteoarthritis changes than any other joints in the body. These changes are often preceded by disc degeneration. In addition to the osteoarthritic changes as a cause of low back pain, the following are possible mechanisms in facet joint pain:

1) Malalignment of the facet joints as a result of narrowing disc space.
2) Strain of the capsular ligaments.
3) Entrapment of a meniscoid in the facet joint.
4) Entrapment of a synovium fringe.

The malalignment and osteoarthritic changes are probably the most common causes of low back pain that originates in the facet joints.

Nerve Root Irritation

The annulus is thinnest at the posterior aspect in the lumbar region. In addition, the posterior longitiudinal ligament tapers in the lumbar region making the postero-lateral aspects of the disc particularlty susceptible to herniation(Fig. I). Disc herniation occurs when the annulus fibrosus is weakened while there is still some turgor in the nucleus pulposus. Thus disc herniations are more frequent in the relatively young patient(peak incidence in the third decade), while it is rare in patients over 60 years of age.

Simple direct pressure on a normal nerve root will not produce the sciatica-like pain. It is probably the inflammatory reaction that causes the pain in nerve root irritation. At surgery the nerve root entrapped by the herniated disc appears red and edematous.

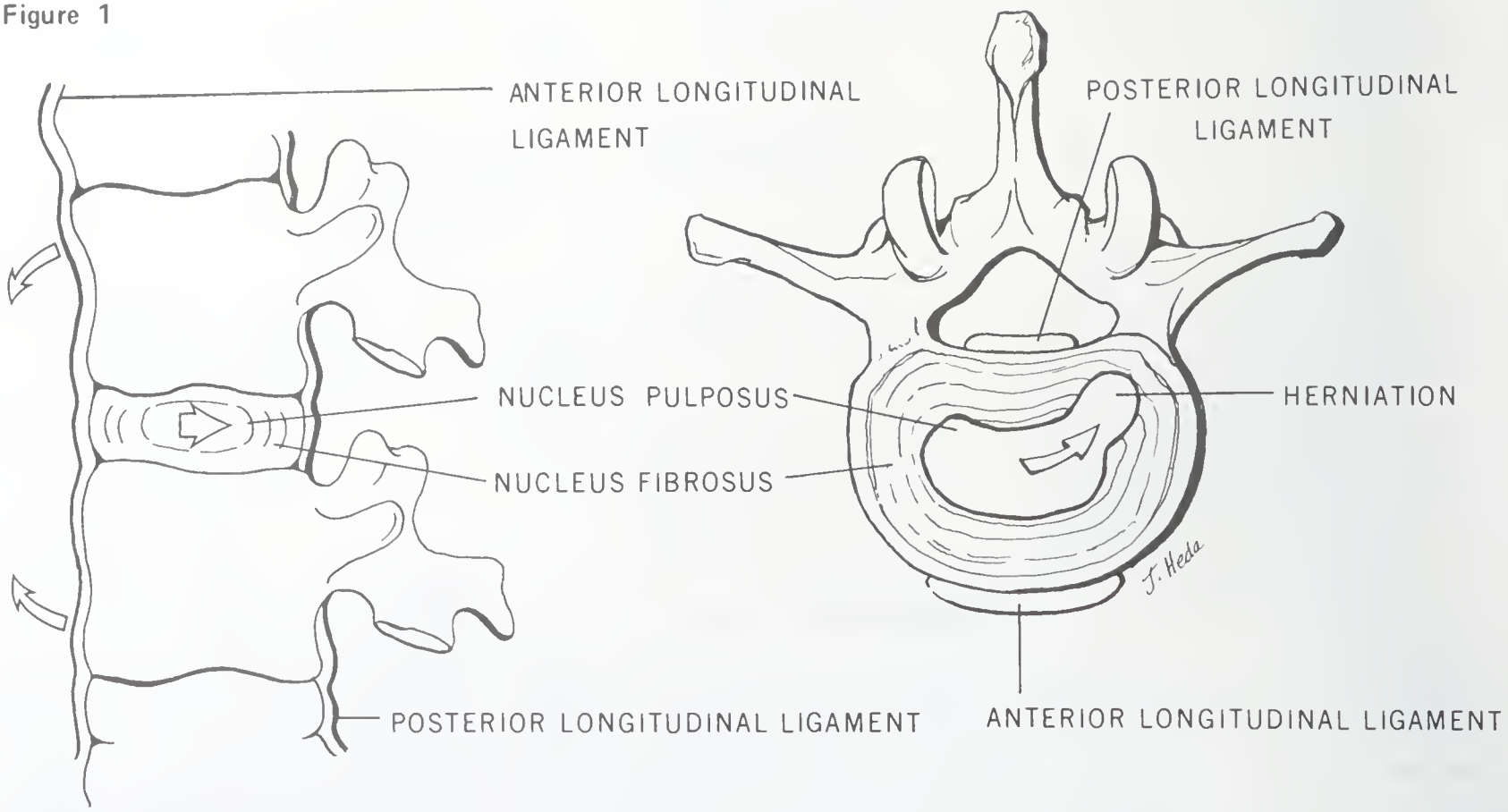
Because the lower lumbar discs are subjected to a greater load and because of the anatomy of the lumbar spine elucidated earlier, about 80 per cent of disc herniations occur at either the L4-L5 level or L5-S1 level giving rise to 5th lumbar or 1st sacral nerve root irritation respectively.

It is to be noted that disc herniation is only one of the mechanisms whereby the nerve root can be entrapped. Osteophytes, ligamentous bands and tumours can all cause nerve root entrapment. Thus it is worthwhile to remember that not all nerve root entrapment is caused by disc herniation and not all disc herniation causes nerve root entrapment.

Clinical Presentation

The age of patients may vary from teens to sixties; the peak incidence and severity occurs in the third and fourth decade of life. The usual explanation for the decrease in incidence of low back pain past the age of sixty is that the narrowed in-

Figure 1



tervertebral joint, having become relatively stiff, is less likely to be painful. In fact, if a patient over the age of 60 presents for the first time with low back pain, one should consider the possibility of systemic or malignant disease.

The clinical picture presented by patients with discogenic or facet joint pain is recurrent low back pain. The majority of these patients have recurrent attacks of pain at intervals ranging from 3 months to three 3 years, each episode lasting from 3 days to 3 months. The patients are relatively pain free between attacks and there is little interference by their back condition in their daily activity.

There are two usual modes of onset: one sudden, the other gradual. The sudden onset of pain occurs during the performance of some trivial action, such as doing up a shoe lace or picking up a book from the floor. The onset of pain is quite dramatic and patients describe their backs as "going out" or "being caught". Often there is a time lag between the precipitating event (many times assumed or not identified) and the development of maximal pain. The delay can be between a few hours to a few days. In low back pain attributable to the facet joint, the onset of pain takes place gradually over a period of one day and the course of the pain usually lasts less than one week. The discogenic pain usually peaks gradually in a week and the course of the pain lasts several weeks.

The recurrence of back pain is occassionally associated with strenuous activities but many patients cannot associate it with any precipitating event. Exertion such as an unusual spurt of weekend sports, gardening, prolonged stooping or bending over and heavy lifting are the types of activities likely to precipitate an exacerbation of low back pain. In general these episodes of low back pain diminish with rest and temporary avoidance of strenuous activities.

A major clue, that the pain is discogenic or facet pain, is that it is frequently increased by maintaining one posture over a long period of time(such as prolonged walking, sitting or standing). Although lying down in a comfortable position temporarily relieves the pain, often the patient will complain of pain on waking in the morning.

Unrelenting and constantly progressive pain over a period of a few months is indicative of more serious underlying pathology, such as neoplasm. Night pain is a characteristic of bone neoplasm. This is in contrast to common types of low back pain which are almost always aggravated by activities and relieved by rest.

Discogenic and facet joint pain is often dull and aching in nature. Paravertebral muscle spasms can also be present. The severity of the pain does not necessarily reflect the amount of degenerative changes. Many people with degenerative changes still remain asymptomatic.

The distribution of pain characteristically has no well-defined border and can be either in the lumbar or sacro-iliac region. Although the diagnosis of sacro-iliac strain has been commonly made in the past, it is now recognised that the sacro-iliac region is a common site for referred pain derived from higher segmental levels. Primary sacro-iliac pathology apart from those following parturition and severe trauma is extremely rare. This is because the sacro-iliac joints have irregular and interlocking surfaces and strong ligaments making it an extremely stable joint.

There are patients who would describe their low back pain as "lumbago" after having been told about this by their doctors or having read about it in popular literature. The term lumbago is best regarded as pain in the lower back: a descriptive term not specifying the cause.

Another term that is often used is "sciatica". In the strictest sense, "sciatica" means pain associated with direct irritation of the sciatic nerve(L4, L5, S1, S2 and S3) and the pain extends mainly down the posterior aspect of the thigh, the lateral aspect of the leg and into the foot. It is important to keep in mind that often pain down the leg associated with low back pain may be referred pain and the patient's complaint of "sciatica" does not necessarily mean that a nerve root is being directly irritated. Many patients complain of pain radiating to the buttocks, anterior thigh and groin, or along the lateral aspect of the thigh to the knee that does not correspond to nerve root distribution. As a general rule, most referred pain is felt above the knee, whereas pain due to direct root irritation may spread to the foot. The pain may be described as aching, crampy or shooting down the legs and may be bilateral. It should be noted that nerve root compression due to disc herniation is usually unilateral.

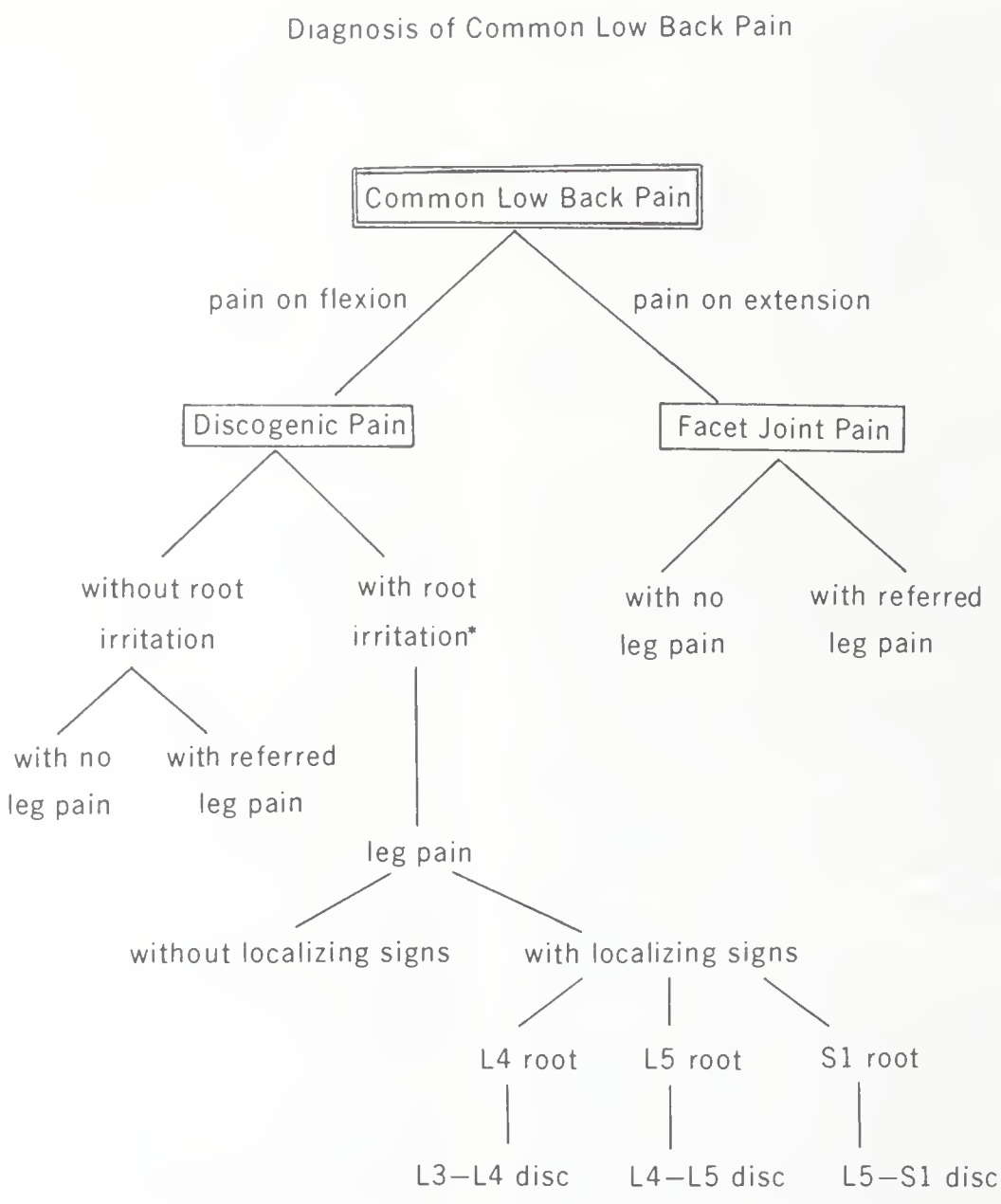
Leg pain that is commonly associated with low back pain that is not due to nerve root compression will not be reproducable by the straight leg raising test.

The differentiation between discogenic and facet joint pain can be made by asking the patient to flex and extend his spine while standing. Facet joint pain is usually aggravated by extension whereas discogenic pain is worse on flexion.

Fig. II, adapted from an article by Dr. Hamilton Hall[2] illustrates the various diagnostic possibilities of the common low back pain syndrome. A patient diagnosed as having only facet joint pain may or may not have associated leg pain. However, one would not, in this case, expect the leg pain to be due to a herniated disc. The discogenic pain also may or may not have associated leg pain and the leg pain may very well be referred pain.

Most discogenic back pain does not have nerve root involement, ie. the straight leg raising test is negative. The patient may or may not have leg pain. If there is leg pain in this case, it is referred pain. In some instances, a diagnosis of a herniated disc is based on the presence of discogenic pain, an associated leg pain and the presence of nerve rooot irritation as demonstrated by a positive straight leg raising test(Fig. II). Although many textbooks give the dermatome distribution of the various nerve roots, it is unusual to be able to localise the level of the lesion simply from the patient's description of the pain distribution. Leg pain is due to an inflammatory process causing nerve root irritation. Not all fibres within the nerve root are irritated to the same degree, and thus the pain may not radiate in a recognisable dermatome distribution.

An irritated nerve root may still conduct nervous implulse normally. In the absence of a conduction deficit, there will be no localizing



*positive straight leg raising test or femoral stretch test

Figure 2

signs in terms of alteration in power, sensation and reflexes. This fact, together with the unreliable distribution of the radiating pain, makes it perfectly acceptable to diagnose discogenic pain with nerve root irritation in the absence of localizing signs(Fig. II). Indeed many people would automatically look very diligently for neurological deficits in the presence of nerve root irritation(ie. a positive straight leg raising test) and would be very confused when none are found. It is perfectly valid to make a diagnosis of discogenic pain with root irritation even when the level of the nerve root compression cannot be localized.

. When a herniated disc causes a conduction impairment in a nerve root, the patient will present with a distinctive cluster of clinical signs. The various combinations of decreased or absent reflexes, sensory deficits, and decreased muscle power enable one to deduce the level of disc involvement. A disc herniation at the L4-L5 level usually affects the L5 nerve root; similarly an L5-S1 disc herniation will compromise the S1 nerve root. The anatomical reason for this is illustrated in Fig. III.

The three most commonly affected discs are at the L4-5, L5-S1 and L3-4 levels in that order of frequency. Many detailed clinical pictures of these commonly affected nerve roots can be found in various textbooks. I found the summary given by Dr. Hamilton Hall to be most useful from a practical point of view(Table I).

An efficient method of physical examination of the back based on a series of positions rather than examining the various systems separately is devised by Dr. Hamilton Hall. This method will not be repeated here and the reader is referred to the original article.[2]

A few salient features regarding the physical examination will be pointed out here. The muscles of the great toe often are the first to show signs of weakness when there is motor involvement. Weakness of the extensor hallucis longus and flexor hallucis longus are found early in motor impairment of L5 and S1 respectively. The sagging of one buttock when the patient stands may indicate that gluteus maximus is weak due to an S1 nerve root lesion. Another point to keep in mind is that muscle wasting is not usually obvious if the motor impairment is less than three weeks in duration and is present in only a small percentage of all root pressure problems.

Regardless of the order in which the various clinical signs are elicited during the physical examination, one should follow a logical sequence of reasoning, as illustrated in Fig. II, in arriving at a satisfactory diagnosis. If one starts at the localizing signs, such as pondering about the significance of an absent ankle reflex, without first considering the other findings in logical sequence, then low back pain will be a very confusing problem. This confusion can come about because of a nerve root compression usually results in a permanent loss of reflexes even though there is complete sensory and motor recovery. Thus the loss of an ankle reflex in the absence of nerve root irritation may merely be a sequela of a previous episode of nerve root compression.

Nerve Stretching Tests

The nerve stretch tests are important in demonstrating nerve root irritation. Inexperience coupled with the lack of anatomical knowledge involved in the tests can often lead to erroneous interpretation of clinical findings.

During elevation of the straight leg with the patient in the supine position, the nerve roots are pulled out of the intervertebral foramina. Normally the roots glide freely through the foramina, but tension is produced when the nerve root is entrapped by a herniated disc.

Tension on the inflammed nerve causes pain in the distribution of the entrapped nerve root. This test is unreliable when the straight leg is elevated beyond 60° for the following two reasons:

1) In the straight leg raising manoeuvre, maximum displacement of the nerve root is achieved at 60° and any pain produced beyond this angle is unlikely to be due to nerve root streching.

2) In individuals with short hamstring muscles, when the straight leg is raised beyond 60°, pain is felt on the posterior aspect of the lower half of the thigh due to tightening of the hamstrings. This is often confused as a positive nerve stretch test.

Whenever the simple straight leg raising test is positive, nerve root irritation should be confirmed by two other supplementary tests(Fig. IV). The straight leg is dropped 10° and the ankle is dorsiflexed. This test is considered positive when the pain is again reproduced. In the bow-string test the popliteal nerve is compressed with the thumb reproducing the pain elicited earlier. The patient's subjective reaction to this test is guaged by squeezing the medial tendons of the hamstring, which should be less painful and the pain should be limited to the area squeezed.

In testing for nerve root irritation asssociated with the femoral nerve(L2, L3 and L4), the patient is tested in the prone position. Flexion at the knee may cause enough stretching of the nerve root to reproduce the pain. Extending the hip will also stretch the femoral nerve. In order for one to say that there is nerve root irritation, the pain elicited in either manoeuvre must radiate in the anterior aspect of the thigh. The mere reproduction of pain in the lower back does not represent nerve root irritation. It is facet joint pain produced by extension of the lumbar vertebra.

A note of warning in doing the nerve streching tests. Rough manipulation of the leg can cause rupture of some axons leading to unnecessary paralysis. This is especially likely to occur during anesthesia when the pain protecting mechanism is absent.

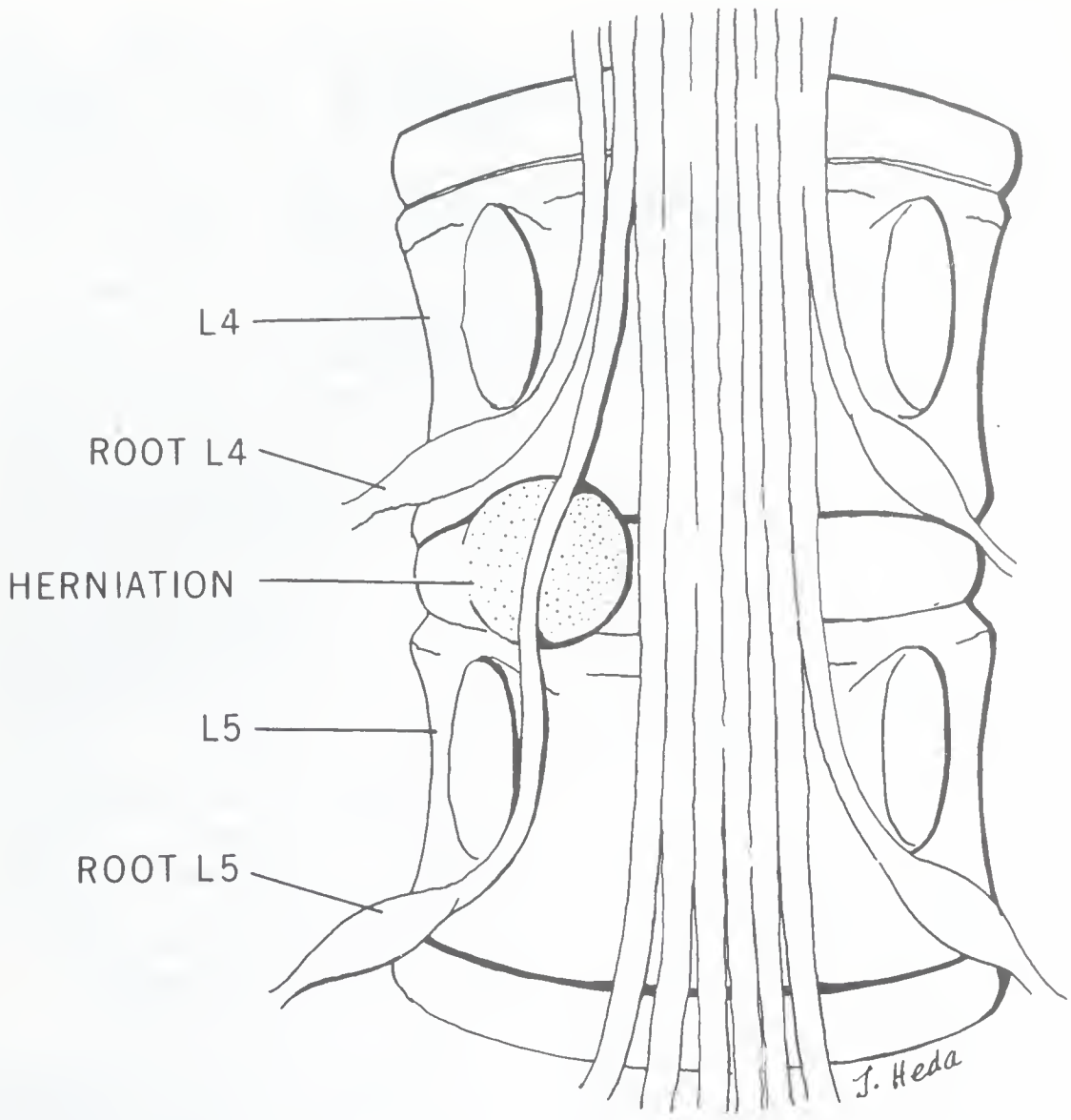


Figure 3

Table 1

Localizing Signs of Commonly Affected Nerve Roots

Disc	Root	Power	Sensation	Reflex
L3—L4	L4	quadriceps	medial calf	knee reflex
L4—L5	L5	foot dorsiflexors hip abductors	lateral calf great toe	nil affected
L5—S1	S1	foot plantar flexors gluteus maximus	5th toe	ankle reflex

X-Ray

X-ray of the lumbar spine is of little if any help in the diagnosis of common low back pain syndrome. Many studies have shown poor correlation between x-ray changes and low back pain. Degenerative changes and anatomical anomalies of the spinal column are frequently seen on x-ray but they do not occur more frequently in the population with low back pain.

The disc being radiolucent, x-ray will not show direct evidence of disc pathology other than a non-specific disc narrowing associated with degenerative changes. In spite of the fact that radiological evidence of degenerative disc disease increases

with increasing age, the incidence of low back pain decreases after the age of 50.

Schmorl's nodes, a common radiological finding, are caused by extrusions of the nuclear material into the spongiosa of the vertebral bodies followed by reactive sclerosis around the extrusions. The only significant point to remember about Schmorl's nodes is that they are not a cause of low back pain.

A disc herniation is never diagnosed on the basis of a plain x-ray as it is not visible radiographically without the introduction of a contrast material. Myelogram is not used routinely to diagnose disc herniation. A myelogram is done only on those cases where one is prepared to act on the findings surgically.

Indications for Surgery

The majority of patients with low back pain, including many with nerve root irritation, can be managed conservatively. After several weeks, the pressure on the nerve is reduced because the herniated portion of the nucleus undergoes fibrosis and shrinks. Thus in most instances surgical decompression is only considered after failure of six weeks of conservative management. An exception to this rule is the presence of severe motor weakness or paralysis early in the course of the disease which indicates severe impingment of the nerve root requiring laminectomy.

Central disc herniation compressing the cauda equina is an indication for emergency surgery. The parasympathetic sacral nerves, which control bladder and bowel functions, being unmyelinated fibres are particularly susceptible to damage by the direct pressure of the herniated disc. Symptoms suggestive of compression of the cauda equina are acute onset of incapacitating low back pain with radiation to both legs associated with bowel and bladder dysfunction. Evidence of bilateral nerve root irritation may be present. The anal sphincter reflex will be impaired. Paresthesia is found in the "saddle area"(S2,3,4). This condition is uncommon but when clinical findings are suggestive an emergency myelogram is indicated.

In summary, the following are indications for surgery:

1) failure of conservative treatment(at least six weeks).
2) increasing neurological deficit in spite of conservative treatment.
3) massive neurological deficit.
4) central disc herniation.

One relative indication is recurrent discogenic pain with significant disruption of socio-economic state.

Summary

Discogenic pain and facet joint pain are common causes of low back pain and often the leg pain associated with it is truly referred pain. Only a small proportion of discogenic pain with leg pain is due to nerve root irritation. An even smaller percentage of these will eventually require surgical decompression after the failure of conservative treatment.

Before any treatment programme for low back pain is initiated, the physician must be confident of his diagnosis. This article has attempted to show a simple and logical approach to the diagnosis of common low back pain.

The author spent a month of elective time with Dr. Hamilton Hall, staff surgeon at Women's College and Toronto General Hospitals and the Director of the Toronto Back Education Unit. The author wishes to thank him for reviewing this article and for his encouragement and teaching.

References

1. MacNab, I., *Backache*. Williams & Wilkins Co., 1977
2. Hall, H., A Logical Approach to the Diagnosis of Back Pain. *Canadian Family Physician*, September, 1975.
3. Finneeson, B.E., *Low Back Pain*. J.B. Lippincott Co., 1972.
4. Fisk, J.W., *A Practical Guide to Management of the Painful Neck and Back*. Charles C. Thomas, 1977.
5. Hastings, D.E., Back Pain, a multifaceted syndrome. *Postgraduate Medicine*, Vol. 62, No. 1, July 1977.
6. Ruge, D. and Wiltse, L.L., Editors, *Spinal Disorder: Diagnosis and Treatment*. Lea & Febiger, 1977.

MEDLINE- Medical Literature On-Line

Bob Heyding

"How many articles have been written on sickle cell anemia in the last two years.?"
"About 475."
"How many of these articles deal specifically with treatment?"
46 of them."
"How many of these 46 articles mention cyanates?"
7 of them."
"Tell me the title and author of two of these 7."
Cyanate-induced cataracts in patients with sickle cell hemoglobinopathies by D.H. Nicholson in Archives of Opthamology 94(6) June 1976 pages 927-30, and Extracorporeal treatment with cyanate in sickle cell disease: preliminary observations in four patients by E.E. Langer in Journal of Laboratory and Clinical Medicine 87(3) March 1976 pages 462-74."

Figure 1: The MEDLINE data base is divided into five smaller data bases each covering two or three years of medical research (approximately 500,000 articles each). The most recent two years can be searched on-line with a computer terminal such as the one above. The earlier data bases are searched off-line and the results are mailed.

This conversation took place between a University of Toronto medical librarian and a computer in Bethesda, Md. It lasted less than four minutes and allowed a U. of T. medical student to quickly identify recent research on the use cyanates in the treatment of sickle cell anemia.

In 1978, over 300,000 articles will be written on medical and medically related topics. In the past five years over 1¼ million articles have been published in more than 3000 medical journals. More has been published in this period than in the first 45 years of this century.

The proliferation of medical literature, the so-called information explosion, has caused problems. University and hospital libraries are finding it increasingly difficult to pay for and store all of the journals that are required for a healthy research atmosphere. The problems of direct concern to the medical student or researcher are:

1) how to select relevant articles from the growing volume
and
2) how to avoid overlooking pertinent material.

The U.S. National Library of Medicine(NLM) publishes a useful index called *Index Medicus* which lists recently published articles under broadly defined subject headings. It

has become an indispensible tool in medical library research. However, because the subject headings do not always accurately reflect the subject content of an article, and because cumulated issues are only published once a year, searching *Index Medicus* can be a tedious and frustrating experience.

In 1972, NLM created *MEDLINE* [*MEDical literature on-LINE*] to supplement and perhaps eventually replace *Index Medicus*. MEDLINE consists of a data base of over 2½ million bibliographic references and an associated on-line interactive information retrieval system. The data base contains references to articles written from 1966 to the present. Each reference includes the title and authors of the article, the journal in which it appeared, the NLM subject headings and (since 1976) a brief summary.

The retrieval system allows anyone with a valid account number to query the data base using on-line computer terminals. In North America, the data base is currently being maintained in NLM in two host computers, one at the State University of New York in Syracuse and the other in Bethesda. Users in Paris, or Toronto, or Tokyo interact with the retrieval system and data base by telephone.

The retrieval system allows users to pick out references that have specified words in the title, that have been assigned specific subject headings, that have been written in a specific language, that have been written by a specific author and so on. It also allows users to combine search terms using the Boolean or logic connectors AND, OR and NOT. For example, the user can ask the system to scan the data base for all articles with both *ANEMIA, SICKLE CELL* as a subject heading AND *cyanate* in the title.

Information systems similar to *MEDLINE* have been developed for many other fields including law, engineering and the natural sciences. It was originally envisaged by people in the information field that these systems would be used directly by students, researchers and practising doctors, lawyers and engineers. However, this has not been the case. Most people who want to search *MEDLINE* obtain the assistance of an intermediary, usually a medical librarian, who is familiar with the searching commands of the retrieval system and the idiosyncracies of NLM's subject headings.

MEDLINE was developed by an American institution for American use. However, NLM has decided to share *MEDLINE* with foreign countries in return for contributed indexing or cash. In Canada, the National Research Council pays $100,000 annually, and distributes *MEDLINE* account numbers on a cost recovery basis to interested

```
Y

PROG:
SS 1  0?
USER:
ANEMIA, SICKLE CELL

PROG:
SS (1) PSTG (475)

SS 2 /C?
USER:
"SUBS APPLY DT, RT, TH

PROG:
SUBHEADINGS ACCEPTED.

SS 2 /C?
USER:
ANEMIA, SICKLE CELL

PROG:
SS (2) PSTG (46)

SS 3 /C?
USER:
"SUBS CANCEL

PROG:
SUBHEADINGS CANCELLED.

SS 3 /C?
USER:
CYANATES OR CYANATE (TW) OR CYANATES (TW)

PROG:
SS (3) PSTG (123)

SS 4 /C?
USER:
2 AND 3

PROG:
SS (4) PSTG (7)

SS 5 /C?
USER:
"PPT

PROG:

1
AU  - CAPPERS J
AU  - CHABRAS A
AU  - DIEDERICH D
TI  - PHYSIOLOGICAL AND CLINICAL IMPLICATIONS OF PROTEIN CARBAMYLATION.
      PP. 501-49.
SO  - N:  GRISOLIA S, ET AL., ED.  THE UREA CYCLE. NEW YORK, WILEY,
      1976.  DU 55 U75 1975. :
```

Medline - Current File
1976 –

DT = Drug Therapy
RT = Radiotherapy
TH = Therapy

```
2
AU  - BREWER GJ
TI  - A VIEW OF THE CURRENT STATUS OF ANTISICKLING THERAPY.
SO  - AM J HEMATOL 1(1):121-8, 1976

3
AU  - ALKJAERSIG N
AU  - FLETCHER A
AU  - JOIST H
AU  - CHAPLIN H JR
TI  - HEMOSTATIC ALTERATIONS ACCOMPANYING SICKLE CELL PAIN CRISES.
SO  - J LAB CLIN MED 88(3):440-9, SEP 76

4
AU  - DEIDERICH DA
AU  - TRUEWORTHY RC
AU  - GILL P
AU  - CRIER AM
AU  - LARSEN WE
TI  - HEMATOLOGIC AND CLINICAL RESPONSES IN PATIENTS WITH SICKLE CELL
      ANEMIA AFTER CHRONIC EXTRACORPOREAL RED CELL CARBAMYLATION.
SO  - J CLIN INVEST 58(3):642-53, SEP 76

5
AU  - TRUBOWITZ S
TI  - THE MANAGEMENT OF SICKLE CELL ANEMIA.
SO  - MED CLIN NORTH AM 60(5):933-44, SEP 76
CONTINUE PRINTING? (YES/NO)

USER:
Y

PROG:

6
AU  - NICHOLSON DH
AU  - HARKNESS JR
AU  - BENSON WE
AU  - PETERSON CM
TI  - CYANATE-INDUCED CATARACTS IN PATIENTS WITH SICKLE-CELL
      HEMOGLOBINOPATHIES.
SO  - ARCH OPHTHALMOL 94(6):927-31, JUN 76

7
AU  - LANGER EE
AU  - STAMATOYANNOPOULOS G
AU  - KLAJTALA MP
AU  - ADAMSON JW
AU  - FIGLEY M
AU  - LABBE RF
AU  - DETTER JC
AU  - FINCH CA
TI  - EXTRACORPOREAL TREATMENT WITH CYANATE IN SICKLE CELL DISEASE:
      PRELIMINARY OBSERVATIONS IN FOUR PATIENTS.
SO  - J LAB CLIN MED 87(3):462-74, MAR 76

SS 5 /C
USER:
"STOP
```

Cost $1.5

Figure 2: A typical MEDLINE search output.

organizations. Most (if not all) Canadian medical school libraries, some hospital libraries, some provincial health departments and a few private companies are now using the system.

Can students afford to use *MEDLINE*? Rates have risen significantly in the last two or three years, but heavy subsidies from the U.S. Department of Health have kept them 30 to 80 per cent lower than rates for similar legal systems. A carefully prepared *MEDLINE* search conducted by a skilled librarian can achieve good results for under $10. Some libraries subsidize *MEDLINE* searches; others charge an additional fee for staff time. The three-minute search at the beginning of this article cost $1.50 at the University of Toronto.

MEDLINE can be operated with a variety of terminals including the television type used by airline ticket agents, the typewriter type used by bank tellers, and the slow speed Telex type. The librarian who is operating the system will telephone either of the host computers in Bethesda or Syracuse) using a low-cost data telephone network. The requestor will supply the words that describe the nature of his request, and the librarian will type these words and/or corresponding subject headings at the terminal. *MEDLINE* will respond to each entry in 5 to 10 seconds with the number of articles that contain the terms or their combinations. In his dialog with the retrieval system, the searcher must be careful to choose words that are neither too broad nor too specific because authors and NLM indexers often use different vocabulary to describe the same concept. This is not a serious problem as many different word combinations can be explored in a short period of time. In fact, interactive flexibility is one of *MEDLINE*'s chief advantages over *Index Medicus*. A normal search takes 10 to 15 minutes.

MEDLINE is an information service with which all medical professionals should become familiar. In 1972 an estimated 25,000 searches were made. In 1978 the number will exceed 800,000.

Footnotes

Greenberg, B., *Medline demand profiles*. Medical Library Association, Bulletin 65, July 1977, p. 65.

Communication in the service of American Health. A Bicentennial Report from the National Library of Medicine. Dept. of Health Education and Welfare Publication Number (NIH) 76-256.

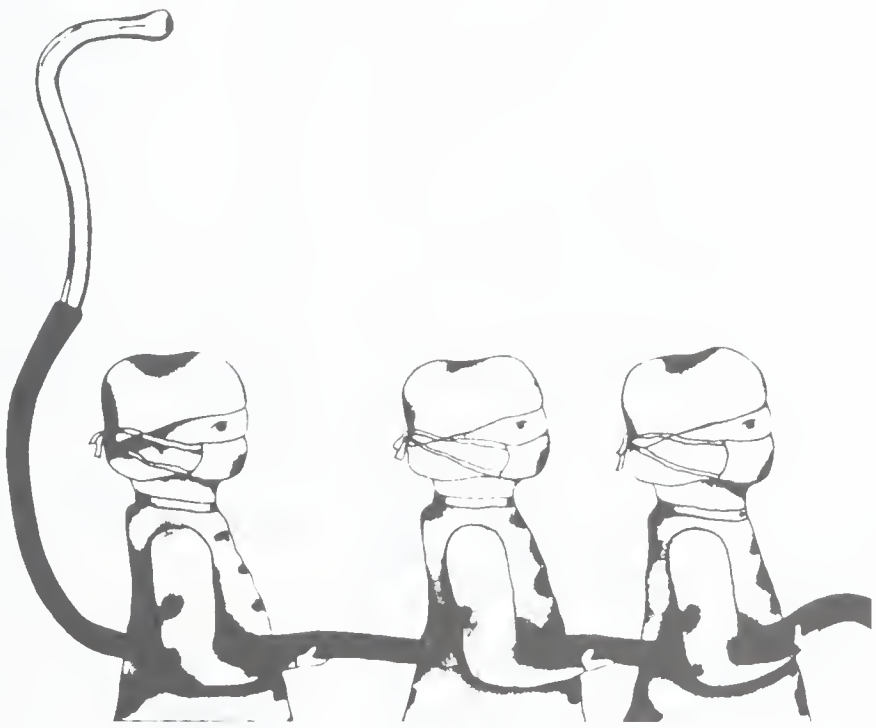
National Library of Medicine News-various issues.

HOW ABOUT YOU?

Nearly 10,000 Medical Students, Interns and Residents have enrolled in

MED-LIFE*

The Exclusive Plan For
MEDICAL STUDENTS
INTERNS and
RESIDENTS



TORONTO CENTRE BRANCH:
E. R. Finn, Manager
101-201 Consumers Road / Phone: 493-4160

TORONTO SCARBORO BRANCH:
Rich A. Bennett, C.L.U. Manager
209-2100 Ellesmere Ave. / Phone: 438-0376

CANADIAN PREMIER LIFE INSURANCE COMPANY

*and predecessor plans.

B7556

Partial Meniscectomy-
A 10 Year Experience

S. Larry Goldenberg & R.W. Jackson, M.D., M.S., F.R.C.S.(C)

The idea of only partially removing a torn meniscus has long been a controversial subject. Early antagonists warned that with partial meniscectomy, one was jeopardizing the regenerative process, as well as possibly overlooking micro-tears [1] which would inevitably cause degenerative problems. Recently, however, several studies have appeared in the orthopaedic literature to suggest that aggressive surgical treatment of a symptomatic knee may lead to more problems than the injury itself. Arthrotomy and meniscectomy is one of the most common procedures performed by orthopaedic surgeons, even though clinical,[2,3] radiographic,[2,4] and arthroscopic[5] studies all have shown that a high percentage of degeneration occurs in post-total meniscectomy knees. But what about the post-partial meniscectomy or "miniscectomy" knees? In 1975, Cox et al [6] compared partial and total meniscectomies in experimental dogs. Their findings, which support earlier work by King[7] (1936) on dogs and Dann[8] (1969) on rabbits, were that degenerative effects were directly proportional to the amount of meniscus removed. In addition, Burke et al[9], (1977) used transducers to study the pressure distribution patterns on the tibial plateaus of human cadaver knees which had been subjected to either partial or total meniscectomy. They showed that "significant load-bearing function of a peripheral rim of meniscus occurs following partial meniscectomy, resulting in a more normal pressure distribution pattern than occurs with a total meniscectomy."

This descriptive study attempts to discover answers to at least two questions in this controversial area:
a) Is there a role for "miniscectomy"?
and
b) Who is the prime candidate?

Method of Study

A total of 69 partial meniscectomies were performed on 65 patients by one of the authors over a 10 year period, between 1966 and 1977. All patients first underwent arthroscopic examination of the injured knee(s) to confirm the diagnosis, to rule out other lesions or "meniscal mimes", and to document the presence or absence of chondromalacia. Where the pathology was suitable, endoscopic surgery was performed; otherwise, arthrotomy was carried out.

At follow-up patients were questioned about their existing symptomatology as compared to their premorbid state, their level of activity, and any interceding problems and/or treatments. Subjective responses were graded according to pre-determined criteria to try and minimize variability, and thus increase validity. (**Table 1**)

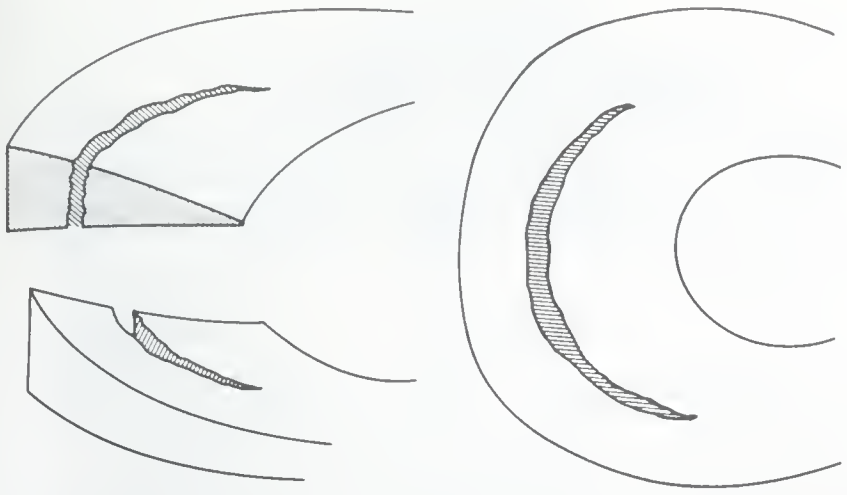
To avoid confusing jargon, lesions are classified according to three basic planes:
1) Longitudinal or vertical (**Figure 1**)
2) Radial or transverse (**Figure 2**)
3) Horizontal or cleavage (**Figure 3**)

If the longitudinal tear remains attached anteriorly and posteriorly, a "bucket-handle" tear results. (B.H.T. - see **Figure 4**) The inner portion of the B.H.T. may become displaced into the intercondylar region and lead to pain and locking on movement.

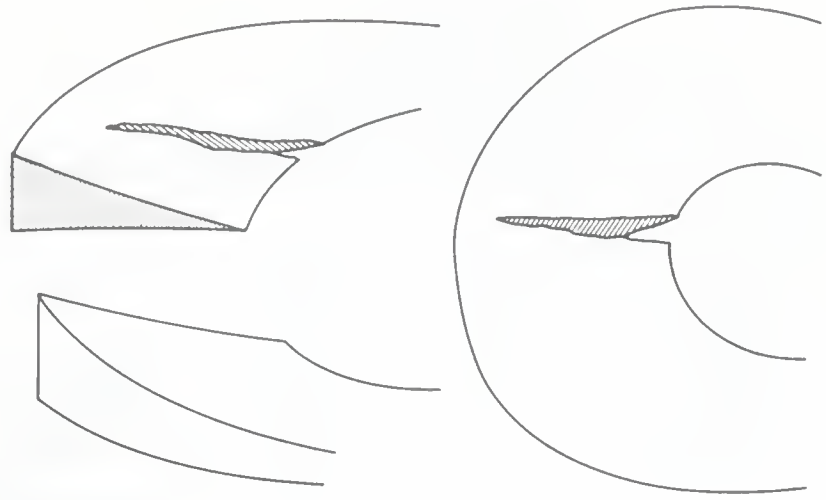
Transverse tears may be small and on the inner border. These more commonly occur toward the anterior horn and result in flaps or tags which often cause a feeling of instability as

TABLE 1

RESULT	CRITERIA
Excellent	Totally asymptomatic; Participates in all sporting activities
Good	Active in rigorous sporting activities (running, squash, skiing, etc.) with a minimal aching or weakness afterwards
Fair	Some disability (pain, weakness) in everyday activities (climbing stairs, squatting) or in cold weather, but remaining active
Poor	No improvement / Severely limited

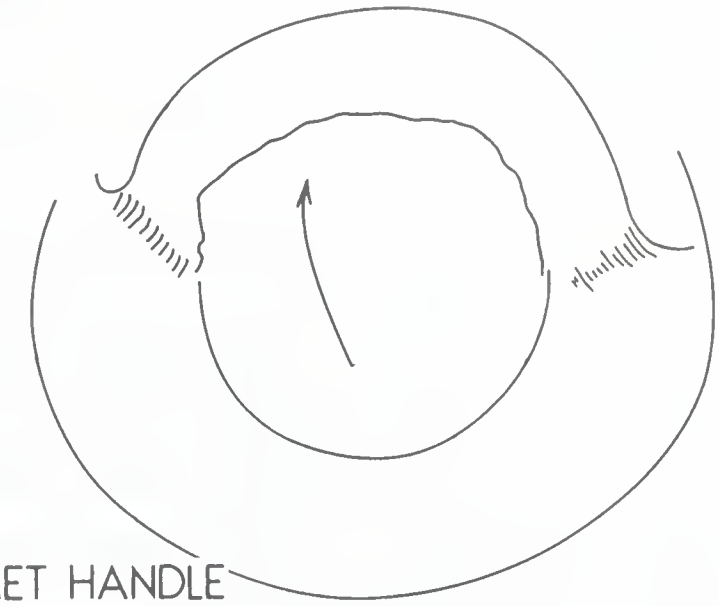


LONGITUDINAL OR VERTICAL



RADIAL OR TRANSVERSE

HORIZONTAL OR CLEAVAGE



BUCKET HANDLE

they enter the weight-bearing area. (**Figure 5**)

Horizontal tears are more common in the posterior horn region and are secondary to shearing stresses. Arthroscopically, they present as a bulging inner border when the knee is flexed and the two torn surfaces slide on one another.

Results

As seen in **Table 2**, 65 patients were studied with a total of 69 knees. Unfortunately, five patients(six knees) were lost to follow-up. There were 54 males and 11 females with a mean age of 31.5 years. Medial meniscii were more commonly involved (72%) than lateral meniscii (28%). Eleven patients (16%) had a second miniscectomy" at the same time or at an earlier date.

Of the 63 miniscectomies reviewed, only one had a "poor"

FLAP

Figures 1 - 5 have been reprinted with the permission of the publishers, C.V. Mosby Company: Arthroscopy and Arthrography of the Knee - A.A.O.S. Instructional Course Lectures, 1978.

TABLE 2

Detail of 65 patients (69 knees)

A) Number lost to follow-up 5 (6 knees)

B) Site of Pathology

	No.	%
Medial	50	72
Lateral	19	28
Both	11	16
Rt. Knee	35	49
Lt. Knee	34	51

C) Age (Years)
Range 15-54 Mean - 31.5

D) Sex
Male 54
Female 11

TABLE 3

ACCUMULATED RESULTS

RESULT	NO.	%	FOLLOW-UP INTERVAL (Range / Mean)	PRE-OP INTERVAL (Range / Mean)
Excellent	47	74.6	2 mo. – 10 yr. (30.6 mos.)	1 day – 12 yrs. (11.8 mos.)
Good	9	14.3	2 mo. – 5 yr. (32 mos.)	3 wks. – 10 yrs. (23.4 mos.)
Fair	6	9.5	4 mo. – 9 yr. (54 mos.)	1 wk. – 10 yrs. (3.1 mos.)
Poor	1	1.6	3.5 yr.	2 yrs.
Total	63	100.0		

result. However, this particular patient was receiving Workman's Compensation benefits and for reasons of secondary gain, ignored medical advice and rehabilitation offers, and eventually developed a fixed flexion contracture. All others had a fair result or better, and a total of 56(88.9%) were much improved as late as 10 years later, participating in many types of competitive sports.

Case Presentation

A 32 year old, nationally-ranked squash player originally injured his right knee in 1962 while playing football. Since then, he had "locking" episodes occurring two or three times a year. Six days prior to admission, the knee locked on the squash court and he could not release it.

Physical examination revealed some tenderness over the lateral aspect of the right knee with loss of 10° of full extension. A diagnosis of a torn lateral meniscus was made and the patient was taken to the operating room.

Through an anterior infero-lateral approach, arthroscopic examination revealed a peripheral longitudinal tear in the lateral meniscus, attached anteriorly, and with the medial portion displaced into the intercondylar notch. Arthrotomy confirmed a true B.H.T. and with a sharp dissection both attachments were divided. The peripheral rim showed two small cleavage tears that were considered insignificant and so the rim was left in to provide stability.

The patient returned to work within three weeks of surgery and was on the squash court again in two months. Over three years later, his knee remains completely normal.

Type of Lesion (Table 4)

The most common lesion in this review was the longitudinal B.H.T., [39 cases, (62%)]. The next most common lesion[20 cases, (32%)] were flaps. In total, 34 of 39 B.H.T.'s(87%) and similarly, 18 of 20 flap tears(90%) had excellent or good results. Medial B.H.T.'s had slightly better results than lateral ones with 77% excellent as compared to 62%. Fair or poor results were more or less evenly distributed with three being medial B.H.T.'s, two lateral B.H.T.'s and two flap tears.

Pre-operative Duration of Symptoms (Table 5)

The duration of symptoms seems to be important with respect to long-term results. One hundred percent of patients seen less than one week after injury have excellent results. Also, the majority of all excellent/good results(88%) were seen within 12 months of injury, with only four patients in this category having a fair or poor outcome. In comparison, 10 patients were operated upon more than one year after the onset of symptoms and of those, three(30%) had fair or poor results.

Age (Table 6)

The distribution of patients with excellent or good results was not significantly different among the various age groups, although those who were 21-29 years of age represent a larger percentage of these results.

Sex (Table 7)

The proportion of males and females with excellent or good results was slightly in favour of the former, having 48 of 53(90%) compared to eight of 10(80%). In addition, when age is considered, it seems that males less than 30 and females over 30 have better results.

Type of Surgery (Table 8)

Only seven of the reviewed cases were done by the "closed method"(i.e.: by endoscopic surgery), but all of these had excellent or good results. In comparison, seven of the open procedures(13%) had a fair or poor result.

Pre-existing Chondromalacia (Table 9)

Arthroscopic diagnosis of chondromalacia was made in 23

TABLE 4

EFFECT OF TYPE OF LESION ON RESULTS

Type of Lesion	Excellent (No.)	Good (No.)	Fair (No.)	Poor (No.)	Total (No.)
a) Bucket Handle					
Medial	24	4	3	0	31
Lateral	5	1	1	1	8
b) Flap Tear					
Medial	12	1	1	0	14
Lateral	4	1	1	0	6
c) Other	2	2			4
Total	**47**	**9**	**6**	**1**	**63**

TABLE 5

PRE–OPERATIVE DURATION OF SYMPTOMS

	Excellent & Good	Fair & Poor	Total
<1 week	6 (10%)	0	6 (10%)
1 wk.–2 mos.	20 (32%)	1 (1%)	21(33%)
3 mos.–1 yr.	23 (36%)	3 (5%)	26(41%)
>1 yr.	7 (11%)	3 (5%)	10(10%)
Total	**56*(89%)**	**7 (11%)**	**63(100%)**

Note: 49 of 56 (88%) were seen within 1 year after injury

patients. Of these, 19(83%) had excellent or good results. This compares with 37 of 40(93%) patients with normal articular cartilage going on to complete recovery.

Side of Involved Knee (Table 10)

Of all medial lesions operated on, 40(90%) had excellent or good results, while 15 of 18 lateral lesions(84%) had the same outcome.

TABLE 6

EFFECT OF AGE ON RESULT

Age	Excellent (No., %)	Excellent & Good (No., %)	Fair & Poor (No., %)	Total (No.)
≤20	8 (17)	10 (18)	1 (14)	11
21-29	17 (30)	21 (38)	3 (43)	24
30-39	11 (23)	12 (21)	0 (0)	12
≥40	11 (24)	13 (23)	3 (43)	16
Total	**47 (100)**	**56 (100)**	**7 (100)**	**63**

Discussion

All patients at follow-up(with the single exception of the one poor result) had an objectively normal knee, on physical examination. Hence, the major difficulty lay in developing subjective criteria from which certain interpretations could be made. Excellent or poor results were well-defined but the good or fair classifications were more variable. However, by asking the patients to compare their follow-up status with their pre-operative symptomatology we believe that our results are real and reproducable. The overall result of 89% in the excellent-good category was well above the results of previous studies on total meniscectomies(**Table 11**).

The duration of pre-operative symptoms seems to be a significant factor and it is urged that a damaged meniscus be treated as early as possible. In addition, the presence of chondromalacia at the time of surgery does not necessarily lead to a poor result as was found in an earlier study on total meniscectomies.[10] Rather, the retention of meniscal tissue probably helps to prevent long-term degeneration.

It must be emphasized that the term "partial" removal of the meniscus means the selective removal of a "bucket-handle" or a "flap" tear, purposefully leaving a significant rim of meniscal tissue. It does *not* mean the unplanned and complete removal of the anterior portion of the meniscus, including the rim, leaving a large posterior horn fragment behind. We feel this latter situation produces an "unbalanced" compartment which can lead to early articular cartilage degeneration and a poor result.

The results support our contention, in that selected instances of meniscal damage, removal of the mobile or unstable segment and retention of as much meniscal tissue as possible, especially a good rim from front to back, can give excellent

TABLES 7–10

EFFECTS OF (a) Sex (b) Type of Surgery (c) Pre-existing Chondromalacia and (d) Side of knee on results

	Excellent (No.,%)			Good (No.,%)			Fair (No.,%)			Poor (No.,%)			Total (No.,%)		
a) Sex															
Male < 30	24		(45)	5		(9)	2		(4)	0			31	53	(100)
> 30	16		(30)	3		(6)	3		(6)	0			22		
Female < 30	1		(10)	1		(10)	1		(10)	1		(10)	4	10	(100)
> 30	6		(60)		0			0		0			6		
													63		
b) Type of Surgery															
Open	41		(73)	8		(14)	6		(11)	1		(2)	56		(100)
Closed	6		(86)	1		(14)		0			0		7		(100)
													63		
c) Pre-existing Chondromalacia															
YES Patellar	5			1			0			1			7		
YES Femoral Condyle	9	16	(70)	2	3	(13)	1	3	(13)	0		(4)	12	23	(100)
YES Both	2			0			2			0			4		
NO	31		(78)	6		(15)	3		(7)	0			40		(100)
													63		
d) Side of Knee															
Medial	34		(77)	6		(14)	4		(9)		0		44		(100)
Lateral	12		(67)	3		(17)	2		(11)	1		(5)	18		(100)
Both	1		(100)	0				0			0		1		(100)
													63		

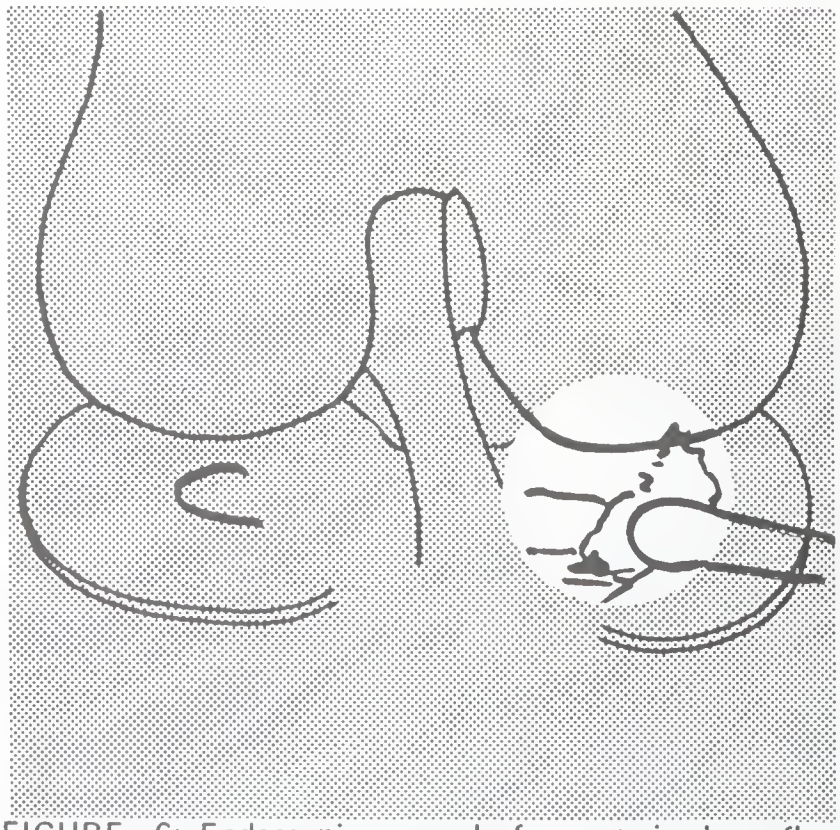


FIGURE 6: Endoscopic removal of an anterior horn flap tear as shown in the accompanying diagram. (Thanks to Grune & Stratton, Inc. N.Y. for their permission)

functional results, and is not bad treatment as frequently taught in the past.

Finally, this study suggests that arthroscopic surgery can lead to a high percentage of excellent results. The numbers involved are small, but nevertheless, the results of endoscopic surgery is certainly no worse than the results obtained by arthrotomy. As the morbidity with endoscopic surgery is so minimal, it would appear to have an increasing role to play in the future.

Conclusions

The aim of partial meniscectomy has been to minimize the amount of surgical trauma and maintain as much function of the joint compartments as possible. The rationale behind leaving a portion of meniscus behind is that it is useful in providing stability, and aids in the lubrication and nourishment of articular cartilage. This has been supported by Cox[11] et al(1977) who found that the presence of a torn meniscus did not lend to degenerative changes in dog's knees unless it interfered with normal function(such as a displaced B.H.T. or free-floating tag). Furthermore, their more severe pathological results(gross and microscopic) were, in fact, produced by total meniscectomies.

Thus, to answer our introductory questions, it seems that partial meniscectomy indeed has an important place in modern orthopaedics. Coupled with the development of first-class arthroscopes and improved techniques of endoscopic surgery, we now have the potential of highly accurate diagnosis, (especially in the acutely injured knee), minimal morbidity following treatment, minimal complications[12], short-stay hospitalization and rapid rehabilitation.

It would appear that the endoscopic removal of a medial B.H.T. in a young male with normal articular cartilage, within 12 months of injury(and preferably within a few days) seems to be an ideal situation. (**Figure 6**)

References

1. Smillie, I.S.: *Injuries of the Knee Joint, 4th. ed.*. Edinburgh, E. & S., Livingstone, 1970, p. 102.
2. Jackson, J.P.: Degenerative Changes in the Knee After Meniscectomy. *JBJS*, 49B, 1967, p. 584.
3. Tapper, E.M. and Hoover, N.W.: *JBJS*, Volume 51-A, No. 3, April 1969, p. 517-526.
4. Appel, H.: Late Results After Meniscectomy in the Knee Joint, A Clinical and Roentogenological Follow-up Investigation. *Acta orthop. Scand. Suppl.*, 133, 1970, p. 1-111.
5. Dandy, D.J. and Jackson, R.W.: The Diagnosis of Problems After Meniscectomy. *JBJS*, Volume 57-B, No. 3, August 1975, p. 349-352.
6. Cox, J.S. et al: The Degenerative Effects of Partial and Total Resection of the Medial Meniscus in Dog's Knees. *Clinical Orthopaedics and Related Research*, No. 109, June 1975, p. 178-183.
7. King, D.: The Function of the Semilunar Cartilages. *JBJS*, 18:1069:1036.
8. Dann, P., Haike, H. and Rosenbauer, K.: der totalen oder partiellen meniscus resektion. *Arch. Orthop. Unfalkhir*, 65:209, 1969.
9. Burke, S.L. et al: A Biochemical Study of Partial and Total Medial Meniscectomy of the Knee. *Trans. of 25th O.R.S.*, February 1978, p. 91.
10. Dandy, D.J. and Jackson, R.W.: Meniscectomy and Chondromalacia of the Femoral Condyle. *JBJS*, Vol. 57-A, No. 8, December 1975, p. 1118.
11. Cox, J.S. and Cordell, L.D.: The Degenerative Effects of Medial Meniscus Tears in Dog's Knees. *Clinical Orthopaedics and Related Research*, No. 125, June 1977, p. 236-241.
12. Jackson, R.W.: The Role of Arthroscopy in the Management of the Arthritic Knee. *Clinical Orthopaedics*, No. 101, June 1974, p. 32.

TABLE 11

TOTAL MENISCECTOMIES	EXCELLENT* –GOOD	FAIR	POOR
R.W. Jackson & D. Dandy	65%	27%	8%
Tapper & Hoover 1968	68%	22%	10%
Lipscome & Henderson 1947	80%	15%	5%
J.R. Huckell 1965	64%		
D.B. Wynn-Parry, P.J.R. Nichols & N.E. Lewis 1958	74%	21%	

* This paper's criteria were adapted to the results of the included reviews.

Thin Air

David Arnold

The veil of oxygen which clothes our planet and gives us life thins dramatically with increasing altitude so that at 5500 metres, a lungful of air contains but half the oxygen it would have at sea level. This observation would be quite unimportant if we all spent our leisure hours dabbling our feet in the waves but every year, increasing numbers of people choose to go to the earth's high places for recreation and since they get there easily and speedily(with minimal acclimation) increasing numbers will suffer in that thin air and suffer altitude sickness. As many as one in twenty persons going above 3050 metres will develop symptoms of altitude sickness and of these perhaps 200 will die each year - needlessly. Dr. Drummond Rennie puts it so well: *"Altitude sickness kills people, and because these are people who go up into the mountains, it kills the young, the fit, the enthusiastic, the audacious and hardworking, and it is killing them in ever increasing numbers..."*

Mountaineers have always had a vital interest in altitude sickness and have learned, albeit the hard way, how to handle it. Today, however, the person standing at 3050m or higher is more than likely a weekend hiker, a deep powder helicopter skier, or even an otherwise sedentary tourist who has just got out of his car to admire the view. Many of these people will need medical aid and some will die in its absence. You may be one of the afflicted yourself.

How will altitude sickness affect you? It might cause headache, weakness, nausea/vomiting, and sleep disturbance. This is acute mountain sickness(AMS). If, in addition you have shortness of breath, cough and stupor you have high altitude pulmonary edema(HAPE). If you have a severe headache, lethargy, stupor progressing to coma you have cerebral edema(CE). HAPE and CE can kill you. Another, usually asymptomatic form of altitude sickness is retinal hemorrage(RH). These forms of altitude illness were known before the turn of the century and were described in a classic 1913 paper by T.H. Ravenhill who was puzzled, among other things, by the observation that persons with 'cardiac puna'(HAPE) showed no evidence of heart disease. The mechanisms of altitude sickness - AMS, HAPE, CE and RH are probably different manifestations of the same pathophysiological process - are still being clarified today. One of the most important research efforts into altitude illness is a joint Canadian-American project called HAPS - high altitude physiology study - based for the past ten summers on Mt. Logan, Canada's highest peak(6035m). I was lucky enough to be part of the 1977 HAPS project.

Mount Logan is a beautiful chunk of rock and ice tucked into the south west corner of the Yukon. It is the

'Logan High' research station at 5335m lying below Mt. Logan's North summit.

highest peak in a group of high mountains called the St. Elias range. Logan rises out of the high surrounding glaciers like a fortress, protected almost on all sides by nearly vertical 3000m to 3500m walls etched with knife-edged ridges. As we flew into Logan from our base camp at Kluane Lake, a hundred breathtaking miles away across icefields, mountains and serpentine glaciers, the mountain certainly looked impregnable but there is one chink in Logan's armour and that was the way we were going. Logan was first climbed by this route in 1925 and when the HAPS project began 10 years ago the mountain had still been climbed only 10 times. Logan seems ready made to be a high altitude research station. Its several summits, all around 5600m to 6035m, surround and slope gently down to a huge virtually flat plateau about 24km long and 10km wide. This plateau is almost miraculously free of crevasses(faults in the imperceptably flowing glacier which can be hundreds of metres deep - a climber's special nightmare) and protected by the peaks around it from the howling winds usually found around mountains. Hence it could be comfortable for a research party and a safe place to land small planes bringing equipment and supplies.

The camp itself is located at 5334m making Logan the highest research station of its kind in the world. For ten years, Charlie Houston, a professor of Medicine at the University of Vermont and probably **the** leading worker in high altitude research as well as being one of the great climbers in mountaineering history, has been leading adventurous, slightly crazy souls and top quality medical researchers(these disparate psyches often inhabit the same body) up to the Yukon to find out what altitude sickness is all about.

This year, there were four areas of research. First, a continuation of studies in exercise physiology at altitude concentrating on alveolar-arterial O_2 gradients. Second, pulmonary mechanics, especially lung volumes and compliance. Third, monitoring of arterial O_2 saturation and respiration during different stages of sleep. (Sleep has been shown to be anything but restful for people with altitude illness. It may, occasionally, be fatal). Fourth, study by retinal photography of the evolution of retinal hemorrhages and measurement of the associated visual field defects. As you can well imagine, our tent containing the apparatus for these experiments looked as sophisticated inside as it looked raw on the outside. The project is organized so that three groups are studied, each in a different stage of acclimation. It was my good fortune to be part of the highly acclimated group. This not only saved me from severe mountain illness(which was experienced by some who were less acclimated) but also allowed me more time in the bracing, rejuvenating and incomparably lovely surroundings of Logan.

We were six, the to-be acclimated group; three men, Fred , Ward and myself, three women, Pauline, Susan and Susan, one family. We came from near and far to McMaster at the end of May. I was near, from Toronto; Pauline was far, from Australia via Alaska.

At McMaster, we were put through the paces to establish low altitude norms for ourselves which would later be compared to our performance at high altitude. We had tubes passed though our noses into our esophagi. Thanks Tony.(Dr. Tony Mansell of HSC). We exercised until we dropped. Thanks John. (Dr. John Sutton of McMaster). We breathed ourselves silly on hypoxic/hypercapnic gas mixtures. (We never figured out who to thank for that). We retched as fluorescein whipped through our veins and into our retinas. "Some people find they get a bit nauseated with fluorescein." Thanks Murry. (Dr. McFadden now at Vancouver General Hospital). They didn't even let us sleep in peace. "You will feel a tiny pricking sensation as each of these 186 electrodes penetrates your scalp." When we were done, we looked like dope addicts. Needle tracks all over the arms, big dilated pupils (neosynephrine, not cocaine), yellow skin (fluorescein, not jaundice) and urine the colour of canaries. In addition we probably looked like escapees from the psychiatric floor at McMaster Medical Centre as we filled taxis with skis, snowshoes and down parkas on that hot and sunny May 29th.

From Hamilton, we flew to the Yukon, to Lake Kluane where our base camp was and thence to Logan where on a sunny June day no-one thought us crazy for having skis, snowshoes and wooly underwear. We climbed slowly and carefully from 3050m to over 5485m before dropping down onto the high plateau at 5335m. The climb took us 12 days, three of which we spent cuddled up in our tents cooking, talking and relaxing while storms raged outside. We went slowly to avoid mountain sickness, about 300m each day. Last year, the group had to evacuate one of their members who developed HAPE; the year before, one climber got cerebral edema and had to go down. We also went slowly because our arterial pO_2's were running about 35 - 45mm Hg. at the higher altitudes and no one feels like trucking along under those conditions. Once on the high plateau, we set about building a camp for the scientists who would come later. They would fly in, virtually unacclimatized, to serve as the acute exposure experimental group. They all got sick to some degree, thus fulfilling a basic tenet of medical research which says "Do it to yourself first".

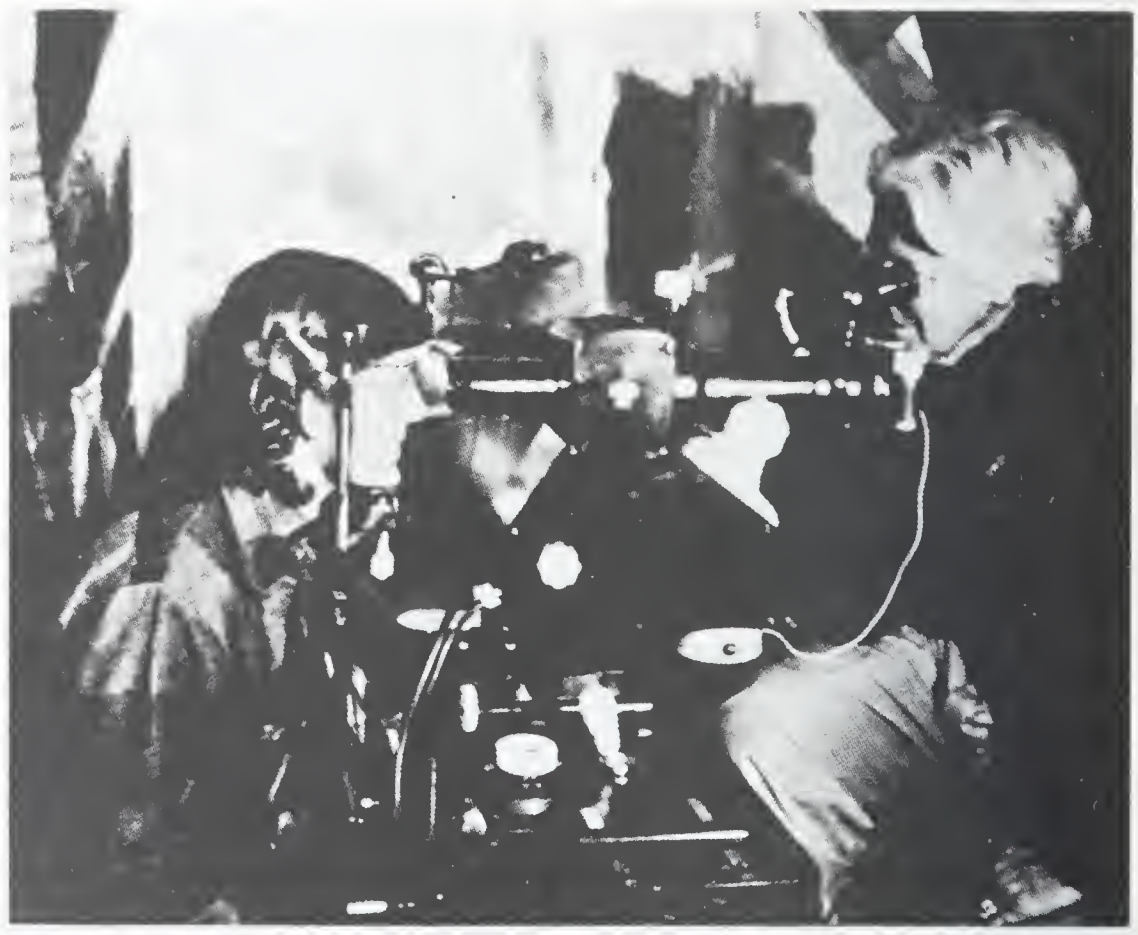
The camp took three weeks to set up, complete with indoor toilets, kitchen, dining room, heated sleeping quarters and electricity. Running water, however, was not possible since the temperature never rose above minus 10° C. All in all, the little huddle of tents and huts aglow with electric lights and warmed with propane heaters seemed somewhat anomalous up among the frozen pristine world where we were. Still, anomalous or not, a few people were thankful we were there. A Japanese climbing party found hot tea and freshly baked cookies waiting for them after a murderous night storm-bound near the summit. A party of five from Oregon were led through a whiteout(where **all** perspective is lost as ground and sky are uniformly white) by the strange and utterly ridiculous(considering where we were) whirring of a power saw. They thought they were hallucinating(q.v. psychiatry - a case of 'folie a cinq').

Once the camp was fully established, the scientific gear all delivered and set up, and the scientists arriving, the research plan went into high gear. Over the next week, we were tested much as we were at McMaster but with the added stresses of altitude. Exercise produced progressive desaturation of our blood as the pO_2 fell from 45mm Hg to values of 35mm or lower(arterial desaturation during exercise never occurs at sea level) probably as a result of either a diffusion problem, venous admixture or both in combination. We found that we could manage only about 66% of our sea level maximums before collapsing. Ward, a 6' 5" giant, fell off the exercise bicycle in a faint while pushing over 200 litres/min. of air into his lungs.

The retinal hemorrhage study showed that almost all of us had RH though in varying degrees of severity. My hemorrhage was a tiny peripheral flame; Susan 's eyes looked like someone had tramped around with hobnail boots. She had multiple hemorrhages and cotton wool exudates where five weeks before her retinas had been spotless. She had multiple visual field scotomata which roughly corresponded with her cotton wool spots. Significantly, perhaps, she had been the member of our party most afflicted with altitude symptoms on the way up. It is rather frightening to think that the kind of vasculopathy and hypoxic injury we could see in her eyes was likely happening unseen in the rest of her body. She is back in California now and her vision is being assessed carefully. Still I should say that retinopathy does not correlate well with the degree of altitude illness and should not be used as the basis of deciding whether or not to evacuate a climber who appears ill on other clinical grounds. Nor do these hemorrhages leave permanent retinal damage as do diabetic and hypertensive retinopathy. The cause of altitude RH is as obscure as for the other forms of mountain sickness.

Several hypotheses were tested this summer but we succeeded only in ruling several out rather than finding a definite causal factor. What is clear is that they are common, rarely serious(though the appearance of the retina may be frightening) and resolve spontaneously over several months. They are clearly part of the whole spectrum of altitude illness but which part remains an enigma. The sleep studies turned up several very interesting observations. One fairly consistent finding was that apparently healthy and clinically normal persons(i.e. me) lapse into periodic breathing once asleep. My particular rhythm was three normal breaths followed by 20-35 second periods of apnea. Apparently you could set your clock by my breathing, it was that regular. What is surprising is that my arterial saturation fell only slightly during these apneic periods from 85% to 70%. How and why breathing is changed remains unknown but it suggests that, as others have already suspected, sleep at altitudes can be lethal. Susan(who also had retinopathy) had her saturation fall to 21% at one point though she averaged about 50%. In fact, whereas most of us had relatively stable saturations throughout the night, she and a few others had wildly swinging values although their respiration appeared entirely regular. We considered instrument error as the cause although the ear oximeters we were using were highly regarded for accuracy; but blood drawn from an indwelling arterial catheter during sleep confirmed the oximeter readings. What is amazing is that after a night where her pO_2 failed to rise above 35mm and fell as low as 15mm she awoke feeling spry and even danced a little jig after getting out of her sleeping bag. Something very curious is going on here! One idea suggests that altitude may uncover or induce a combination of disco-ordinate respiration and sensitivity to hypoxia which resembles in a gross kind of way the problems with sleeping infants who die from crib death. This combination allows progressive hypoventilation to go unnoticed by the body until the brain and heart become fatally hypoxic. With her 21%, Susan won the desaturation contest but it was a close battle with Dr. John Sutton from McMaster who reached a personal low of 29%. Many of us were glad we weren't in the running.

The full results of the summer's research will be published this spring and might bring the enigma of mountain sickness closer to solution. And an enigma it truly is. The physiology and pathophysiology of adaptation to altitude is too complex



Retinal photography helps in the detection and monitoring of retinal hemorrhages which occur in at least 33% of climbers at high altitude.

for me to go into here. Still, the facts as known about HAPE suggest that the basic pathological mechanisms are peculiar. I quote from an article in *The Lancet*(Nov. 27, 1976) by Drummond Rennie, an ex-Loganeer:

"The facts about high-altitude pulmonary edema seem to be as follows. It is much commoner in children than in adults. It is related to severe exercise and is worse at night when oxygen desaturation is greatest. Like all forms of acute mountain sickness it is related to the speed of the ascent being much commoner in those who have flown to high altitudes. Patients with HAPE get, on ascent, an excessive arterial desaturation with a raised alveolar/arterial oxygen gradient uncorrected by 100% oxygen. They also show, on ascent, an abnormally great, and increasing rise in pulmonary artery pressure, which occurs early, before edema, with electrocardiographic evidence of acute right heart strain. Left atrial pressure is normal as are pulmonary capillary wedge pressures, so there is no evidence for pulmonary venoconstriction, just as there is no evidence for myocardial dysfunction and no venous distension. Interstitial edema probably preceeds frank alveolar pulmonary edema at altitude[West et al have suggested that interstitial edema causes increased pulmonary vascular resistance with pre-capillary vessel closure]. In some cases fatal widespread vascular thromboses have been found.

There are virtually as many theories for HAPE as there are investigators in the field. Fortunately, despite the ambiguity about mechanisms, there is no question as to therapy though the efficacy of the therapy only adds to the mystery. A child could tell you that if you get sick by going up fast, you should come down fast. This is the definitive therapeutic intervention. Oxygen, morphine, furosemide, steroids are all ancillary and should never take the place of a controlled but rapid descent. A climber(or skier) who is obtundated and incoherent at 5200m will often be totally lucid two hours later at 4300m. How ascent causes and descent reverses altitude illness lies at the heart of understanding mountain sickness. Hypoxia alone fails to explain all the changes which occur.

While research into the mechanisms of AMS, HAPE, CE and RH goes on, there still some basic and very important points which anyone, medically trained or not can learn about mountain sickness:

1. How to prevent it.
2. How to recognize it.
3. How to treat it.

Prevention is simple. People should ascend slowly when going above 2000m; the suggested rate is 300m each day and if you go higher during the day, you should return to a lower altitude to sleep. Prior conditioning has **no effect** on preventing any of the forms of mountain sickness; paradoxically, in fact, it often seems to be the most fitness concious person in the party who becomes ill. Acetazolamide (Diamox), a weak diuretic, taken for several days **prior** to ascending has been found to reduce the incidence of AMS though by predictably obscure mechanisms.

Recognition is simple. Anyone unduly weak, fatigued or short of breath may be developing HAPE. A dry cough is usually present. Eventually obvious rales and pink-tinged sputum will be present. Ataxia and headache are usually the first symptoms of CE which often co-exists with HAPE. The ataxia is often too easily attributed to bad boots, slippery snow or muscle fatigue. Arm and hand clumsiness is much less common. Eventually hallucinations may occur and the level of conciousness deteriorates towards coma.

Treatment is simple. Anyone with these symptoms must be taken down immediately. Waiting to see if things get worse is folly; trying to evacuate a semi-comatose climber through deep and crevassed snow will endanger the entire party. Oxygen, diuretics and steroids may help if administered properly but none are as effective as a thousand metres of descent, which is almost always curative. You must resist any selfish desires to go on or selfless refusal to go down by the sick person. It is under these dual circumstances that deaths from altitude illness occur and every one of these deaths is preventable. Remember these few simple instructions and even if you never understand the pathogenesis of altitude sickness, you'll administer sound, sensible and effective care to people who become ill at altitude.

Social Policy for Mental Health in Ontario

Bruce G. Pollock

In our last issue Mr. Pollock traced the evolution of social policy for mental health in this province from the early years of the century up to the 1954 Mental Health Act. We are pleased to present herein the conclusion of his article: a brief, yet reflective consideration of the 1967 Mental Health Act and the most recently proposed Amendments thereto.

The Mental Health Act, 1967

The Mental Health Act of 1954 was an important step in the development of a comprehensive social policy pertaining to mental health. Involving the community health and welfare services in an integrated approach to comprehensive community care, it initiated closer linkages between the mental hospital and the community.

The new Mental Health Act passed in 1967, went a great deal further to strengthen these linkages. Following the precedents established in Britain and Saskatchewan, it passed an "umbrella statute" embracing all the mental health facilities of the province into what it was hoped would be a comprehensive system of care. All in-patient facilities were now required to include out-patient, day care, and emergency services, and consultative and educational services to community agencies.

An important new aim was to provide mental health care on the same basis as the provision of health care in general. This concern was rooted in the premise of the Tyhurst Report prepared by the Canadian Mental Health Association that:

> The patient suffering from mental illness should receive the same excellence of medical and ancillary health services as quickly, easily and efficiently as the patient suffering from physical illness.

Thus a paramount feature of the new Mental Health Act was its authorization for "informal" admission, whereby anyone believed to require the observation, care and treatment provided in a psychiatric facility might be admitted on the recommendation of a physician, in a fashion similar to general hospital admission. No loss of personal or civil rights was to result from the the patient's signed consent for treatment.

The Committee on Legislation and Psychiatric Disorder of the Canadian Mental Health Association, in its study on the rights of the mentally ill, supported this innovation in its conviction that:

> The admission to psychiatric facilities of patients who are willing to take treatment should be on the same basis as admission of other ill people to hospitals or clinics.

This policy superceded the proviso for "voluntary" admission, which was declared to be "little more than a self-commitment."

Also in keeping with the policy of modelling mental hospital legislation on general hospital practices, was the repeal of the procedure of trial discharge, or probation. Like the discharge policy adhered to in general hospitals, a patient released in the community was now to be considered officially discharged. Moreover, now that certification procedures were dispensed with, re-admission to hospital, if necessary, would not be so onerous and stigmatizing a procedure. There would not be so great a tendency, either, for community agencies to refer a patient back to hospital when confronted with re-adjustment problems as when he was on probationary status.

Patient's Rights

The new Act's concern with equalizing mental health care with treatment for physical illness was part and parcel of its objective to preserve the dignity and rights of the mentally ill patient. This legislative landmark has been hailed as a "Bill of Rights" for the mentally ill.

Although "informal" admission was adhered to whenever possible, provision was also made for the eventuality that involuntary committal to hospital might be necessary. Yet the only requirement for this latter procedure was the signed application of one physician. Due regards for the rights and freedom of the individual was sustained, as evidenced in review board legislation and the stipulation for involuntary committal only when deemed absolutely necessary in the interests of the patient's "own safety or the safety of others."

This measure was in accordance with the conviction of the World Health Organization that:

> It must be possible to subject certain patients who are either dangerous or unaware of their state to treatment without their consent.

No longer would medical certificates serve as legal authorization to detain patients for indeterminate lengths of time in the mental hospitals of the province. The attending physician was henceforth legally bound to reconsider thoroughly the cases of detained patients. The intervals at which these re-examinations were to be perfomed were clearly specified, and were "calculated to reflect sound medical practice, and to protect the civil rights of the individual."

Review Boards

In keeping with the Act's concern for the preservation of the rights of the mentally ill, an involuntary patient was eligi ble to apply for a review of his case each time his detention was renewed by the attending physician. When a hearing was held by the review board established for this purpose, the patient himself could attend the hearing, unless otherwise directed by the chairman, in which case he could send a representative.

Review boards had jurisdiction over all psychiatric facilities in which patients were hospitalized involuntarily. They were organized on a regional basis, with each board consisting of psychiatric, legal and lay membership; the hospital was required to furnish the review board with any required "information and reports" for examination and conducting of the review itself.

Underlying the establishment of review boards was the rationale that

> In order to permit the simplified and expeditious practice of requiring only one physician to participate in certification, it is necessary to build in a device for the protection of civil liberty.

The review board was found to be workable both in the United Kingdom and in several parts of Canada. It was felt that since the board met in the hospital, it was easier for the patient to appear without the stigma of public exposue of his problem in court. Moreover, the board could act more quickly and was a readily "seen" protection for the patient.

The Present

The policy set forth in the 1967 Mental Health Act has laid the groundwork for the development of provincial mental health services up to the present. One might question the extent to which the enlightened measures established in the Act have been implemented in services to meet the varied needs throughout the province. There is an increasing outcry against custodial care in the large impersonal institutions and for the development of such innovative community services as the Gender Identity Clinic at the Clarke Institute of Psychiatry.

Yet one cannot help but suspect that our politicians, in their zeal to close hospitals with the promise of an increase in community services, are goaded as much(if not more) by present-day inflationary pressures as by a concern for effective patient care.

Moreover, it would be naive, as Dr. Jan Dukszta, M.P.P. and former practicing psychiatrist has pointed out, to think that differently trained personnel such as social workers, doctors or psychologists have learned, as yet, to communicate effectively. It is also obvious that there is still a shortage of and lack of co-ordination between the community mental health services; all too many people are still doing without the help they need.

The 1967 Mental Health Act seems to have sparked the stimulus for ongoing concern with patient care, criteria for involuntary committal and the rights of the mentally ill. It is obvious that the Act cannot be expected to provide the ultimate solution to all the problematic complexities indigenous to mental illness and the promotion of mental health. Amendments to the Act have recently been introduced in the Ontario Legislature which reflect present concerns. [1]

These concerns are replete with controversy--healthy controversy, in my view, revolving around what is considered to be in the patient's best interests. The very concept of mental illness as a criterion of "safety" to self or others in the 1967 Act to "bodily harm" in the proposed legislative amendments.[2] Psychiatrists, on the other hand, who perceive mental illness within a much broader context, are concerned with the many seriously ill individuals who require hospitalization but do not pose a threat with regard to "bodily harm".[3]

The length of involuntary committal is another serious bone of contention. The Civil Rights activists want this time period reduced from the month stipulated within the 1967 Act to the 72 hours proposed in the new legislation.[4] Many physicians, on the other hand, feel that this time period is too restrictive for purposes of obtaining an adequate assessment.[5] Several other issues percolating within the new Bill before the Legislature are provoking further conflict such as the legalistic and/or medical perspective on mental illness for purposes of institutional confinement; confidentiality of patient information; competency to manage one's own affairs--issues too numerous and complex to discuss at this juncture.

It is my own concern that the 1967 Act is coming to be perceived as an instrument for forced confinement and the abrogation of the patient's rights rather than as a humane, practicable policy for mental health care, the raison d'etre for its establishment. Hospitals were never intended as places of imprisonment within the context of this Act but, rather, as therapeutic resources.

The very controversy that the Ontario Mental Health Act of 1967 has engendered at the present time attests to its power as a catalytic agent for dynamic change within the mental health system throughout the province. In the last analysis, it has strengthened the ties between hospital and community and, more importantly, between professional, layman and patient in the betterment of mental health care for all.

References

1. Ontario, *An Act to amend The Mental Health Act*, Bill 124, December 13, 1977.
2. *Ibid.*, sec. 3.
3. Ontario Medical Association, *Press Statement*, March 7, 1978.
4. Ontario, *Bill 124*, sec. 5.
5. O.M.A., *op. cit.*

With this final issue of Vol. 55 the Editor wishes to inform our subscribers and advertisers that next year the Journal will undergo a major change in format, as determined by a vote of the U. of T. Medical Assembly March 20, 1978. The traditional five issues per Volume will be trimmed to three.

Best wishes for a successful Vol. 56 to Lynn McIntyre, 8T0, next year's Editor, and many thanks to the loyal and hard-working Staff of Vol. 55, our friends in the Department of Arts as Applied to Medicine, and our printers, Visual Media Productions and Webcom, Ltd.

Susan Dickinson Ross, 7T9
Editor

Educational Resources available from the

Addiction Research Foundation

—books—

Social Aspects of the Medical Use of Psychotropic Drugs
October 1974 179 pages **$6.50**
Editor: Ruth Cooperstock

Alcoholic Liver Pathology
January 1975 369 pages **$17.00**
Editor: J.M. Khanna Ph.D., *et al*

Clinical Pharmacology and Toxicology of Psychoactive Drugs
March 1975 228 pages **$10.00**
Editor: E.M. Sellers M.D., Ph.D., FRACP

Biological and Behavioral Approaches to Drug Dependence
April 1975 192 pages **$6.50**
Editors: Howard D. Cappell, Ph.D., A.E. LeBlanc, Ph.D.

Alcohol, Drugs, and Brain Damage
June 1975 136 pages **$5.50**
Editor: J. Rankin MB., FRACP

—videocassettes—

¾" U-Matic

Detoxication: The Ontario Model
November 1974 25 Min. Color **$100.00**

Antabuse: A Second Chance for Choosing
April 1975 21 Min. Color **$95.00**

Contracting — A Therapists Aid to Family Counselling
June 1975 47 Min. Color **$95.00**

Tranquillizers: The Popular Panacea
Patterns of Prescription drug use in Canada
September 1975 20 Min. Color **$95.00**

Helping Skills — A Demonstration of Robert Carkhuff's Model
February 1976 53 Min. Color **$80.00**

Moderation — At All Times?
March 1977 5 Min. Color **$80.00**

For more titles and information enquire at:

Marketing Department
Addiction Research Foundation
33 Russell Street
Toronto, Ontario, Canada M5S 2S1

Telephone **416-595-6056**